Revictimization as a Moderator of Psychosocial Risk Factors for Problem Drinking in Female Sexual Assault Survivors*

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ABSTRACT. Objective: Adult sexual assault (ASA) survivors report greater levels of problem drinking than do other women, and research suggests that their coping strategies, reactions from their social networks, and traumatic life events affect their problem drinking. The links between these factors and problem drinking may be moderated by whether survivors are revictimized, yet research has not examined this possibility. Therefore, the current study examined psychosocial factors, problem drinking, and revictimization in women ASA survivors. **Method:** Community-dwelling urban women (n = 555) who had experienced an ASA completed a mail survey at Time 1 (T1) and were resurveyed 1 year later to examine how revictimization between survey waves moderated the effects of coping strategies, social reactions to assault disclosures, and traumatic life events on problem drinking at Time 2 (T2). **Results:** The findings showed that recent revictimization that occurred between surveys was related to increased problem drinking at T2, after T1 problem drinking was controlled for. Moderated hierarchical multiple regressions showed that survivors who engaged in drinking to cope with distress, who received negative social reactions in response to recent assault disclosures, or who experienced additional traumatic events had increased T2 problem drinking only if they were revictimized since T1. **Conclusions:** Psychosocial factors relate to increases in problem drinking for sexually revictimized women but not for nonrevictimized women. Interventions to reduce problem drinking in women ASA survivors should target drinking to cope with assault-related symptomatology, informal social networks to improve their supportiveness, and safety issues through risk-reduction education and self-defense training for women when appropriate. (*J. Stud. Alcohol Drugs* **70:** 41-49)

FEMALE ADULT SEXUAL ASSAULT (ASA) survivors experience greater levels of problem drinking than women who have not experienced ASA (Burnam et al., 1988; Kilpatrick et al., 1997; Wilsnack et al., 1997). Increased problem drinking among ASA survivors may result from the strategies these women engage in to cope with their victimization experiences. In general, avoidance coping strategies (e.g., withdrawal, denial) relate to poorer outcomes in ASA survivors, including problem drinking (Frazier et al., 2005; Santello and Leitenberg, 1993; Ullman et al., 2007a; Valentiner et al., 1996). Few studies, however, have examined the separate effects of drinking to cope with distress and other avoidance coping strategies in relation to problem drinking in women or ASA survivors. Recent cross-sectional studies of women ASA survivors suggest that avoidance coping is associated with posttraumatic stress disorder (PTSD) symptoms (Ullman et al., 2007a), whereas drinking to cope with distress relates to problem drinking (Ullman et al., 2005). Thus, drinking to cope with distress should be examined specifically, because that response to ASA may be most critical in influencing changes in problem drinking.

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In addition to survivors' own behaviors and responses to victimization, it is important to consider the effects of reactions received when survivors disclose the ASA to their social network members. Unfortunately, social network members and formal support sources frequently have negative responses (e.g., blaming) to survivors' ASA disclosures (Ullman, 1999). Such negative responses can have a harmful impact on women problem drinkers—for example, by increasing the likelihood of relapse after treatment (McCrady and Epstein, 2005). Alcohol problems may occur when survivors use maladaptive strategies such as drinking to cope with distress resulting from negative reactions.

Cumulative trauma exposure, both sexual and nonsexual, poses risks for many aspects of health (Schnurr and Green, 2004), including problem drinking. In addition, more chronic traumatic stressors such as child sexual abuse (CSA) are commonly associated with problem drinking in adults (Ouimette and Brown, 2003). Cross-sectional research has shown that drinking to cope with distress and expectations that alcohol would reduce tension, but not PTSD symptoms, mediated the effects of distal trauma histories on current problem drinking in women ASA survivors (Ullman et al., 2005). Also, women with histories of sexual victimization (e.g., CSA) are more likely to engage specifically in drinking to cope, possibly in response to PTSD symptoms (Bissonnette et al., 1997; Hussey and Singer, 1993). Thus, problem drinking in ASA survivors may be a product of complex interrelations between trauma histories, psychological sequelae

(e.g., PTSD, depression), and the specific coping strategies in which survivors engage (e.g., general avoidance coping, drinking to cope with distress).

Revictimization as a moderator

Women who experience multiple ASAs may be at even greater risk for negative outcomes, including increased problem drinking (Kilpatrick et al., 1997). Revictimization may reignite negative sequelae from earlier assault experiences (e.g., Nishith et al., 2000), which may activate maladaptive coping strategies, leading to problem drinking. Research has yet to examine, however, whether general avoidance coping or drinking to cope with distress relate to problem drinking more for women with recent victimization, compared with women with only more distal ASA experiences. Thus, the present longitudinal study examined whether sexual revictimization moderated the effects of these coping strategies on women ASA survivors' problem drinking over 1 year. We predicted that the effects of avoidance coping and drinking to cope on future problem drinking would be greater for women who were revictimized during the study, compared with women who were not.

Revictimization also may moderate the relations between negative reactions and problem drinking, because revictimized survivors may be more likely to feel that their repeated assault experiences signify that the victimization is their fault, especially if they are blamed and treated poorly by others (Ullman, 1997; Ullman et al., 2007b). To the extent that revictimized survivors experience these feelings more than women who are not revictimized, they may be more likely to develop alcohol problems as they attempt to cope with associated distress. Therefore, we expected that the effect of receiving negative social reactions to disclosures of assaults reported at Time 1 (T1) on problem drinking at Time 2 (T2) would be moderated by sexual revictimization.

Similarly, recent sexual victimization may reignite effects of past traumatic events and exacerbate maladaptive coping responses to them, thereby increasing the risk of negative outcomes (Aldwin, 2007; Aldwin and Yancura, 2004), including problem drinking. Past research shows cumulative effects of sexual victimization and other traumatic events on worse outcomes in ASA survivors (Leitenberg et al., 2004; Matheson et al., 2007; Ullman and Brecklin, 2002), but the effects of prior traumas actually may be magnified by revictimization. ASA survivors who experience sexual victimization again may be particularly distressed by this additional trauma, and research suggests that revictimized women suffer worse outcomes than nonrevictimized women, including problem drinking (Kilpatrick et al., 1997). One reason for this finding may be that sexual victimization is more strongly related to PTSD than other traumatic events (Kessler et al., 1995). Thus, to the extent that revictimization aggravates previous PTSD or other psychological symptoms experienced as a consequence of other traumas, and survivors self-medicate with alcohol to deal with symptoms, we expected that traumatic events would be associated with greater problem drinking in women who experienced recent sexual victimization than in women who did not.

In summary, the effects of psychosocial variables such as coping strategies, negative reactions to assault disclosures, and other traumatic experiences on problem drinking may be moderated by whether ASA survivors experience further sexual victimization. Little research has examined how psychosocial factors and traumatic life events relate to changes in women survivors' problem drinking, however, nor have studies examined whether revictimization moderates these relations. Therefore, longitudinal studies are needed including all of these factors to better understand risk factors related to ASA survivors' problem drinking. We predicted that T1 measures of avoidance coping, drinking to cope, negative social reactions, and other traumatic events would each be associated with greater problem drinking in women and that these effects would be moderated by recent sexual victimization such that the strength of the associations would be greater for women who were revictimized than for women who were not.

Method

Participants and procedure

The present longitudinal study analyzed data from a large, diverse sample of ASA survivors who completed two self-report mail surveys administered 1 year apart. Advertisements in local newspapers and flyers distributed throughout Chicago area college campuses, community sites (e.g., bookstores), mental health agencies, and rape crisis centers invited women ages 18 or older with unwanted sexual experiences since age 14 to participate in a confidential mail survey. Interested women callers were mailed the initial survey (i.e., T1) with a cover letter and information sheet describing the study, a list of community resources for women survivors of violence, and a postcard to return if they were interested in participating in the 1-year follow-up survey (i.e., T2). Women provided informed consent before participation and received $20 for completing the T1 survey. Of those women who requested the initial survey, 1,084 returned it—a 90% response rate. Most women (n = 909) who completed the T1 survey expressed an interest in completing the follow-up survey. Approximately 1 year after they completed the T1 survey, participants were mailed the second survey with an up-to-date community resource sheet. Women also were paid $20 for completion of the T2 survey. T2 surveys were returned by 625 women—a 69% response rate. Including only respondents who reported an ASA at T1 (N = 969), we compared women who completed only our

T1 survey (n = 414) with those who completed both the T1 and T2 surveys (n = 555): They did not differ significantly on any study or demographic variables (all t's = -1.87–1.74, 728–963 df, p's ≥ .06; all χ^2's ≤ 7.75, 1–7 df, p's ≥ .15, n's = 955–969). Our final sample for the purposes of this article included 555 women who reported an ASA at T1 and who completed both waves of data collection. All participants were treated in accordance with the ethical guidelines of the University of Illinois at Chicago.

Measures

Sample demographics. At T1, participants reported their age (mean [SD] = 33 [11]), education (14% completed less than 12th grade, 13% graduated high school, and 72% had at least some college), school status (27% were students), employment status (51% were employed), sexual orientation (75% were heterosexual, 7% were lesbian, 13% were bisexual, and 5% were unsure), marital status (57% were single, 28% were married/cohabitating, 14% were separated/divorced, and 1% were widowed), parental status (43% had children), ethnicity (38% were white, 45% were black, 6% were Hispanic, 2% were Asian, and 7% were of other ethnicities), and household income (36% earned $10,000 or less, 54% earned between $10,001 and $50,000, and 9% earned more than $50,000).

Adult sexual assault measure. ASA at age 14 years or older was assessed dichotomously (yes/no) using the Sexual Experiences Survey (SES; Koss et al., 1987). The SES has reported internal consistency reliability of .69 and test-retest reliability at 1 week apart of 93% (Koss and Gidycz, 1985). This sample includes only women who reported an experience of ASA at T1 (76% experienced completed rape, 10% experienced attempted rape, 10% experienced sexual coercion, and 4% experienced unwanted sexual contact). Twenty-nine percent of the women reported that they were drinking before the assault (yes/no). The women were on average 21 (7) years old at the time of the assault, and they reported that their ASAs occurred an average of 13 (11) years ago. Revictimization was assessed with the SES on the T2 survey, identifying 245 women (45% of the sample) who had an ASA experience since the last survey (yes/no).

Coping strategies. Participants completed the Brief COPE at T1, a 28-item scale of coping strategies (Carver et al., 1989). Various coping strategies used in the past 30 days to cope with the assault were assessed with Likert items ranging from 1 ("I didn't do this at all") to 4 ("I did this a lot"). The COPE has been used widely in studies of stressed populations and has adequate internal consistency reliability (all but one subscale $\alpha \geq .60$) and test-retest reliability (r's = .46–.86). A composite measure of reliance on avoidance coping strategies was computed as the unweighted sum of responses to six items comprising the behavioral disengagement, denial, and self-distraction subscales of the COPE (α = .72; mean = 11.97 [4.06]). Similarly, a composite measure of reliance on substance-use coping strategies was computed from two items assessing whether participants used alcohol or drugs to make themselves "feel better" or to help them "get through it" (α = .93; mean = 3.75 [2.26]).

Social reactions to assault disclosure. The Social Reactions Questionnaire (SRQ; Ullman, 2000) assessed 48 different social reactions ever received from persons to whom survivors had disclosed their ASAs (five ordinal categories from "never" to "always"). The mean number of negative social reactions (taking control of the victim's decisions, blaming the victim, treating the victim differently/stigma, distraction/discouraging talking, egocentric responses) was computed (mean = 1.01 [0.66]). The SRQ has good test-retest reliability (r's = .68–.77; Ullman, 2000). The SRQ was administered again at T2, adapted so that women reported social reactions received since the last survey in response to disclosures of the initial ASA (mean = 0.86 [0.72]).

Lifetime history of traumatic events. Goodman et al.'s (1998) Stressful Life Events Screening Questionnaire (SLESQ) assessed 10 traumatic events of an interpersonal nature (i.e., a life-threatening illness or accident, physical abuse as a child or adult, a robbery or mugging involving physical force or a weapon, being otherwise threatened with a weapon, someone close suffering a violent death, witnessing interpersonal violence, serious injury or life being in danger, or some other extremely frightening or horrifying situation). This measure was scored as the summed number of events experienced by each respondent, excluding ASA and CSA (mean = 3.08 [2.08]). If respondents reported the same incident under more than one item, it was counted as one event. The SLESQ has good test-retest reliability (median κ = .73) and adequate convergent validity with a lengthier interview (median κ = .64). The SLESQ was re-administered at T2, adapted so that women were asked to report traumatic life experiences they had had since the last survey (mean = 0.87 [1.24]).

CSA measure. Following Koss et al. (1987), the SES was administered at T1 with respect to whether women had experienced CSA before age 14 years (yes/no). Fifty-four percent of the sample reported having experienced CSA, 44% of whom experienced completed rape, 17% attempted rape, 25% sexual coercion, and 14% unwanted sexual contact.

PTSD symptom severity. We used the Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995), a standardized 17-item instrument, to assess the total number of PTSD symptoms women reported experiencing. At T1, women rated how often each symptom had bothered them in relation to the ASA during the past 30 days on a scale ranging from 0 ("not at all") to 3 ("almost always"). Validated with ASA survivors (Foa et al., 1997), the PDS has acceptable test-retest reliability (κ = .74) for a PTSD diagnosis over a 2-week

interval, 87% agreement, and a Pearson *r* of .83 between two administrations. The PDS also has demonstrated good internal consistency (α = .92) and adequate convergent validity (κ = .59) with the Structured Clinical Interview for the DSM-IV Axis I Disorders (SCID-I) PTSD module, indicating 79% agreement between the two measures (Foa et al., 1997). The measure also was reliable in our sample (Cronbach's α = .92). We computed a PTSD symptom severity score (range: 0–51) by summing response weights to individual items corresponding to re-experiencing, numbing/avoidance, and arousal criteria symptoms (mean = 19.06 [12.38]).

Depression. Depression was measured with a shortened, 10-item version of the Center for Epidemiologic Studies-Depression Scale (CESD-10; Andresen et al., 1994). Participants responded according to how often they felt as described in each item (e.g., lonely, sad) during the past week on a scale ranging from 0 ("rarely or none of the time") to 3 ("most or all of the time"). A mean score indicated the average frequency of respondents' depressive symptoms (mean = 1.58 [0.70]). Cronbach's α of the CESD was found to be .85 in the general population and .90 for patients. The content, concurrent, and discriminant validity have been supported (Radloff, 1977; Weissman et al., 1977). The CESD-10 correlated highly with the 20-item scale (*r* = .97; Andresen et al., 1994).

Problem drinking. The Michigan Alcoholism Screening Test (MAST; Selzer, 1971), a widely used, 25-item standardized screening instrument for alcohol abuse and dependence, was used to assess problem drinking. The MAST has good internal-consistency reliability with a psychiatric outpatient sample (α = .91; Zung, 1980) and has been used successfully in other nonclinical studies of women's problem drinking (Shields et al., 2007). Past-year problem drinking was assessed at T1, and participants responded to the measure at T2 based on their drinking experiences since the last survey. Problem drinking was coded as a continuous measure of total alcohol problems based on Selzer's (1971) guidelines. Thus, certain items were weighted more strongly than others (e.g., "drinking fairly often before noon" was weighted as "1," whereas "a drunk driving arrest" was weighted as "2"; T1: mean = 5.89 [8.59]; T2: mean = 5.96 [9.03]).

Results

A series of moderated hierarchical multiple regression analyses was conducted to examine our prediction that revictimization would moderate the effects of drinking to cope, general avoidance coping, negative reactions, and traumatic life events on T2 problem drinking. We included several control variables in our analyses. First, we controlled for T1 problem drinking, because previous problem drinking is likely to be the strongest predictor of subsequent problem drinking. A history of CSA and T1 traumatic life events was controlled to remove variance in problem drinking associated with distal traumas. We also controlled both T1 and T2 negative social reactions, because past research suggests that negative social interactions affect the risk of relapse in women alcoholics in treatment (McCrady and Epstein, 2005). We controlled for PTSD and depression because of known relations between these variables and problem drinking (Hesselbrock and Hesselbrock, 1997; Stewart and Israeli, 2002). Because alcohol-related ASAs may be more common for problem drinkers because of their greater likelihood of being in risky situations that involve drinking (Parks and Miller, 1997; Ullman, 2003) and because survivors of these kinds of ASAs may be at greater risk for increased problem drinking, we controlled for this assault-related variable. Finally, previous research has found strong relations between drinking to cope with distress and problem drinking (Carpenter and Hasin, 1999; Cooper et al., 1995); therefore, we controlled for T1 drinking to cope. Controlling for these variables makes our analyses more conservative; that is, any significant interactive effects are above and beyond those that might otherwise be expected (e.g., drinking due to depression or PTSD).

To control for nonessential multicollinearity, variables not on a ratio scale (i.e., drinking to cope, avoidance coping, negative reactions) were centered. There was no evidence of multicollinearity (variance inflation factors ranged from 1.18 to 2.04 with all variables entered), and assumptions of linearity, normality, and homoscedasticity of residuals were met for all variables. The assumption of homogeneous error variances was violated for each model; however. Aguinis et al. (1999) recommended using Alexander's *A* (a normalized *t* approximation that is more robust to deviations from normality) when error variances for subgroup slope differences are heterogeneous. Thus, we report both *A* and *F* values for all interaction terms. If these statistics converge, we can be confident that revictimization significantly moderates the relations between predictors and T2 problem drinking (Aguinis et al., 1999).

Predictor variables were entered in three steps. In the first step, CSA history and T1 measures of problem drinking, traumatic life events, alcohol-related assault, PTSD, depression, negative reactions, and drinking to cope were entered into the equation. T2 negative reactions and revictimization were entered in the second step. Finally, interaction terms were entered into the equation in the third step. T2 problem drinking was not significantly correlated with alcohol-related assault (*r* = .04, 374 df, NS), but it was significantly correlated with each of the other predictor variables (*r*'s $\geq$ .15, 212–390 df's, *p*'s $\leq$ .003). The full correlation matrix is available on request. Table 1 shows standardized regression coefficients (β) and *t* statistics for the final step of each model. Results of models showing all steps are available on request.

TABLE 1. Final step of moderated multiple regressions testing interactions of revictimization and psychosocial factors on T2 problem drinking

	Model 1		Model 2		Model 3		Model 4	
Predictors	β	*t*	β	*t*	β	*t*	β	*t*
T1 problem drinking	0.47‡	7.25	0.47‡	7.00	0.47‡	7.02	0.45‡	7.04
CSA history	-0.03	-0.50	-0.02	-0.32	-0.03	-0.39	-0.01	-0.14
T1 traumatic life events	0.02	0.31	0.00	0.06	0.00	0.01	-0.03	-0.45
T1 alcohol-related assault	0.01	0.10	-0.01	-0.18	-0.03	-0.47	0.00	0.04
T1 PTSD	-0.08	-1.10	-0.08	-1.08	-0.09	-1.21	-0.09	-1.24
T1 depression	0.04	0.68	0.06	0.86	0.07	1.00	0.03	0.55
T1 negative reactions	-0.01	-0.12	0.01	0.11	0.03	0.38	-0.01	-0.10
T1 drinking to cope	-0.05	-0.51	0.18†	2.50	0.17*	2.45	0.14*	2.08
T1 avoidance coping	0.06	0.78	-0.08	-0.77	0.07	0.85	0.04	0.60
Revictimization	0.12§	1.88	0.13*	2.11	0.17†	2.55	0.01	0.19
T2 negative reactions	0.20†	2.70	0.23†	3.05	-0.00	-0.03	0.12	1.58
T2 traumatic life events	–[a]	–	–	–	–	–	0.07	0.51
Revictimization × Psychosocial Factor	0.33‡	3.92	0.18§	1.94	0.25*	1.98	0.30§	1.93

Notes: Model 1 examined interactive effect of revictimization and Time 1 (T1) drinking to cope; Model 2 examined interactive effect of revictimization and T1 avoidance coping; Model 3 examined interactive effect of revictimization and T2 negative reactions; Model 4 examined interactive effect of revictimization and Time 2 (T2) traumatic life events. CSA = childhood sexual abuse; PTSD = posttraumatic stress disorder. [a]A dash (–) signifies that the factor was not included in the model.
§$p \leq .10$; *$p \leq .05$; †$p \leq .01$; ‡$p \leq .001$.

Model 1: Revictimization and T1 drinking to cope

T1 variables accounted for 48% of the variance in T2 problem drinking ($R^2 = .48$; $F = 14.74$, 9/142 df, $p < .001$). Even after the positive relation between T1 and T2 problem drinking is controlled for, the tendency to drink as a coping mechanism was significantly and positively associated with increased problem drinking as measured 1 year later. No other T1 variables entered in the first step significantly influenced T2 problem drinking. T2 negative reactions and revictimization were both positively related to T2 problem drinking, accounting for an additional 7% of variance ($R^2 = .55$; $R^2_{\Delta\Delta} = .07$; $F_{\Delta\Delta} = 10.16$, 2/140 df, $p < .001$). The interaction of revictimization and T1 drinking to cope accounted for an additional 5% of variance in T2 problem drinking ($R^2 = .59$; $R^2_{\Delta\Delta} = .05$; $F_{\Delta\Delta} = 15.36$, 1/139 df, $p < .001$; $A = 19.27$, $p < .001$).

To understand this interaction further, we examined the simple slopes of revictimization. Supporting our hypothesis that the relations between T1 drinking to cope and T2 problem drinking would be different for women who were and were not revictimized, T1 drinking to cope was significantly and positively associated with increased levels of T2 problem drinking in women who were revictimized between surveys (β = .30; $t = 4.52$, 139 df, $p < .001$) but not in women who were not revictimized (β = -.03; $t = -.51$, 139 df, NS). After the interaction term was entered in the final step, T1 drinking to cope no longer accounted for unique variance in T2 problem drinking.

Model 2: Revictimization and T1 avoidance coping

The results of Steps 1 and 2 of this model were the same as for Model 1. Although general avoidance coping was not a significant predictor of T2 problem drinking, the interaction of revictimization and T1 avoidance coping accounted for 1% of the variance in T2 problem drinking ($R^2_{\Delta} = .01$; $F_{\Delta} = 3.74$, 1/139 df, $p = .06$; $A = 9.47$, $p = .002$), but the effect was only marginally significant. We tested the simple slopes of revictimization, but neither slope was significant.

Model 3: Revictimization and T2 negative reactions

We also tested whether revictimization moderated the effects of T1 negative reactions and T1 traumatic life events on T2 problem drinking, but neither interaction was significant (results available on request). To examine whether *recent* rather than distal experiences are more important predictors of problem drinking, we tested moderated models with revictimization and T2 negative reactions and T2 traumatic life events. Both models included all of the predictors used in previous analyses.

The results of Steps 1 and 2 of the model examining the interactive effect of revictimization and T2 negative reactions on T2 problem drinking were the same as for Models 1 and 2. The interaction of revictimization and T2 negative reactions accounted for an additional 1% of variance in T2 problem drinking ($R^2_{\Delta} = .01$; $F_{\Delta} = 3.92$, 1/139 df, $p = .05$; $A = 8.07$, $p = .005$).

We examined the simple slopes of revictimization to further understand this interaction. Consistent with our hypothesis, T2 negative reactions were significantly and positively associated with increased levels of T2 problem drinking for women who were revictimized between surveys (β = .24; $t = 3.61$, 139 df, $p < .001$) but not for nonrevictimized women (β = -.00, $t = -.03$, 139 df, NS). After the interaction term was entered in the final step, T2 negative reactions did not account for unique variance in T2 problem drinking.

Model 4: Revictimization and T2 traumatic life events

Step 1 of this model was identical to that in the previously described models. T2 negative reactions, revictimization, and T2 traumatic life events, when entered in the second step, accounted for an additional 13% of the variance in T2 problem drinking (R^2_Δ = .13; F_Δ = 15.23, 3/135 df, p < .001). T2 traumatic life events were positively related to T2 problem drinking. However, T2 negative reactions were only marginally related to T2 problem drinking, and revictimization did not have a significant effect on T2 problem drinking. The interaction of revictimization and T2 traumatic life events accounted for an additional 1% of the variance in T2 problem drinking (R^2_Δ = .01; F_Δ = 3.72, 1/134 df, p = .06; A = 15.84, p < .001).

To further understand this marginally significant interaction, we tested the simple slopes of revictimization. T2 traumatic life events did not predict T2 problem drinking for nonrevictimized participants (β = .03; t = .51, 134 df, NS). T2 traumatic life events were significantly and positively associated with increased levels of T2 problem drinking for revictimized participants only (β = .37; t = 4.88, 134 df, p < .001), as hypothesized, confirming that distal traumas are less important than proximal traumas in predicting problem drinking over time. Finally, after the interaction term was entered in the final step, T2 traumatic life events were no longer significantly associated with T2 problem drinking.

Summary

CSA history, traumatic life events, PTSD, depression, T1 negative social reactions, and whether the assault involved alcohol did not significantly affect problem drinking across the models tested. In contrast, even after controlling for survivors' trauma histories, psychological distress, and prior problem drinking at T1, the tendency to drink as a coping mechanism was a significant factor in predicting participants' problem drinking 1 year later. T2 negative reactions also exerted some significant main effects on T2 problem drinking. As hypothesized, T2 problem drinking was greater for participants who were revictimized, and revictimization interacted with T1 drinking to cope, T2 negative reactions, and T2 traumatic life events to predict problem drinking. This pattern demonstrates that drinking to cope and recent negative reactions and traumatic life events had a significant impact on problem drinking for women who experienced revictimization between surveys but not for nonrevictimized women.

Discussion

Researchers have not examined how the psychosocial factors studied here (e.g., coping, social network responses) influence changes in women's problem drinking using multivariate analyses in community-residing ASA survivors. The present study is unique because it provides evidence that further sexual victimization interacts with psychosocial risk factors to contribute to increased problem drinking in ASA survivors. Specifically, we found that revictimization moderated the negative effects of (1) drinking to cope with distress and (2) recent negative social reactions and traumatic life events on problem drinking over 1 year in a sample of women ASA survivors. Drinking to cope and recent negative reactions or traumas appear to act in concert with revictimization to contribute to subsequent problem drinking. Past cross-sectional studies showed that these variables were related to more problem drinking in this sample (Ullman et al., 2005; Ullman et al., 2008). These variables, however, were associated with subsequent increased problem drinking only for women who were revictimized during our longitudinal study, even after previous problem drinking and other associated sequelae (e.g., depression, PTSD) were controlled for. Although we cannot determine the exact sequence of effects because we have only two waves of data, the results of this study suggest that psychosocial factors may relate differently to problem drinking for revictimized than for nonrevictimized women.

Survivors who engaged in drinking to cope with distress reported significantly increased T2 problem drinking if they had been revictimized since T1, whereas this effect was not significant for general avoidance coping. This finding may reflect that drinking to cope with distress leads to greater risk of sexual revictimization, perhaps because of risky contexts such as bars or parties where women may consume alcohol (Parks and Miller, 1997). In fact, in our study, revictimization was associated with both T1 drinking to cope (r = .13, $p \leq .01$) and T1 problem drinking (r = .20, $p \leq .001$). Revictimized women may be more predisposed to greater use of this coping strategy, which may in turn relate to increased problem drinking. Given that drinking to cope with distress is related to greater problem drinking in general population samples, particularly in women (Holahan et al., 2001; Park and Levenson, 2002; r = .38, $p \leq .001$, in our sample), this coping strategy requires further inquiry to fully understand the relations between revictimization and women's problem drinking.

Unpublished qualitative data have revealed that some women in our sample said that they engaged in increased drinking and/or other substance use to numb the pain associated with repeated sexual victimization. Such a pattern is consistent with self-medication models of problem drinking (Cappell and Greeley, 1987; Stewart and Israeli, 2002), as well as studies of patients in substance-use treatment, which show that PTSD symptoms precipitate substance use to cope, which then increases the severity of substance-use problems (Stewart and Conrod, 2003). Because revictimization is likely to be associated with increased PTSD symptoms (Arata, 2002; Ullman and Brecklin, 2002), these survivors

may be more likely to continue or increase their use of alcohol or other substances following additional victimization incidents.

Despite having significant zero-order correlations, however, T1 psychological symptoms (i.e., depression, PTSD) were unrelated to problem drinking in our models. Further, the null effects of T1 psychological symptoms are consistent with previous cross-sectional research showing no association between PTSD and problem drinking (Ullman et al., 2005). Also, a more recent study by Testa et al. (2007) showed that, after prior heavy drinking and demographic variables were controlled for, PTSD did not mediate the prospective effect of ASA on heavy drinking. This finding suggests the need for further research aimed at understanding how specific coping strategies relate to problem drinking in samples of ASA survivors. That other forms of avoidance coping (e.g., mental or behavioral disengagement) were only marginally associated with T2 problem drinking suggests that drinking to cope may be particularly important in understanding problem drinking and may be particularly sensitive to revictimization.

Surprisingly, several other T1 variables (i.e., CSA history, negative social reactions, traumatic life events, and whether the ASA was alcohol related) that have predicted problem drinking in prior research (e.g., Hesselbrock and Hesselbrock, 1997; McCrady and Epstein, 2005; Stewart and Israeli, 2002) did not increase problem drinking over time in our study, regardless of whether women were sexually revictimized. This may simply be because drinking to cope and T1 problem drinking were much stronger correlates of T2 problem drinking. Many of the ASAs assessed at T1 happened long ago. Therefore, characteristics of those assaults may be less consequential for women's drinking than their other sexual and nonsexual traumatic experiences. Still, alcohol-related ASAs may be important to examine, because recent research shows that some assault-related variables relate to increased revictimization risk (Macy et al., 2006).

In contrast, recent experiences appear to play a stronger role than do past experiences in predicting problem drinking over time. For example, traumatic events experienced during the course of the study, but not lifetime histories of traumatic events, were associated with increased T2 problem drinking for revictimized survivors only. This result is consistent with Kilpatrick et al.'s (1998) finding that women who were assaulted during their longitudinal study were more likely to develop alcohol dependence at follow-up than their nonassaulted counterparts. This finding reinforces the importance of considering proximal traumas to understand problem drinking, as well as the cumulative effect of sexual and nonsexual traumas on changes in problem drinking over time.

Finally, the results of the present study suggest that negative social reactions may lead to more negative outcomes (e.g., problem drinking) for women who have experienced repeated sexual victimizations, compared with nonrevictimized women. Multiple victimization experiences may be associated with greater stigma and worse problem drinking in women. Being blamed for a past sexual assault (these reactions were asked about with respect to the T1 ASA) may lead to greater distress and PTSD, as cross-sectional data reported elsewhere have shown (Ullman et al., 2007a). However, being blamed in combination with being revictimized may make women feel doubly ashamed and could contribute to increased problem drinking. Furthermore, revictimized women may be more likely to seek support from formal sources, which are more likely to respond with negative reactions (Starzynski et al., 2005), in turn possibly increasing problem drinking over the long term. Yet, it also is possible that effects of negative reactions on current problem drinking are temporal and subside as time passes.

This study was limited by having only two waves of data collected from survivors who varied at baseline in time since they had been sexually assaulted. This large sample was diverse in terms of ethnicity and socioeconomic status, however, and it included survivors who can be difficult to reach with traditional random sampling methods (e.g., women without telephones, in shelters). In addition, many survivors also had significant histories of other traumas and experienced a new ASA over the 1-year study period. Despite variation among women in this nonrepresentative community sample, psychosocial factors associated with problem drinking in past cross-sectional work predicted problem drinking after 1 year and were moderated by revictimization status such that the psychosocial factors predicted problem drinking for revictimized women but not for their nonrevictimized counterparts.

The use of the MAST as our only measure of problem drinking is another study limitation. A meta-analysis conducted by Shields and colleagues (2007) suggested that the MAST may not be a reliable measure of problem drinking, especially in samples like ours (i.e., women, nonclinical). Problems associated with this measure may stem partially from the fact that it relies on respondents' self-reports. Because MAST items are face valid, women may not have answered honestly regarding the extent of their problem drinking. Also, women's self-reports on this measure may have reflected distress rather than actual problem drinking.

Understanding changes in ASA survivors' problem drinking may be facilitated by looking at their revictimization risk, recent traumatic life events, drinking to cope with distress, and social reactions of their support networks to ASA disclosures. General avoidance coping appears to be less important, although this factor is important for other consequences of assault (e.g., self-blame, PTSD; Ullman et al., 2007b). The results highlight the importance of understanding the effects of drinking to cope on assault-related symptoms, which supports self-medication theory (Cappell and Greeley, 1987; Stewart and Israeli, 2002). Yet, the results

also imply that theories of women's problem drinking should account for repeated victimization and social network experiences (e.g., negative reactions). These findings are consistent with the broader literature on coping, social support, and drinking, which shows the importance of avoidant forms of coping for predicting women's problem drinking over time (Holahan et al., 2001), as well as the crucial role of negative social support in women's relapse risk after treatment for drinking problems (McCrady and Epstein, 2005).

Repeated sexual victimization and its effect on psychosocial factors known to be related to problem drinking warrant further attention in research on the epidemiology of women's problem drinking and interventions to reduce the problem (Miller and Mancuso, 2004). Such interventions should target drinking to cope with distress, efforts to improve the supportiveness of informal social networks, and safety issues through risk-reduction education and self-defense training when appropriate.

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